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Vocal communication of simulated pain

Jordan Raine^{1*}, Katarzyna Pisanski¹, Julia Simner², David Reby¹

Mammal Vocal Communication and Cognition Research Group, School of Psychology,
University of Sussex, Brighton, UK

Synaesthesia and Sensory Integration Lab, School of Psychology, University of Sussex,
Brighton, UK

Corresponding author contact details:

Address: School of Psychology, Room 2A13, Pevensey Building, University of Sussex,
Falmer, BN1 9QH

Phone: +44 (0)1273 876638

Email: jordan@raineonline.co.uk

Author contact details:

Katarzyna Pisanski: k.pisanski@sussex.ac.uk

Julia Simner: j.simner@sussex.ac.uk

David Reby: reby@sussex.ac.uk

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While evidence suggests that pain cries produced by human babies and other mammal infants communicate acoustic cues to pain intensity, whether the pain vocalisations of human adults also encode pain intensity, and which acoustic characteristics influence listeners' perceptions, remains unexplored. Here, we investigated how trained actors communicated pain by comparing the acoustic characteristics of nonverbal vocalisations expressing different levels of pain intensity (mild, moderate, and severe). We then performed playback experiments to examine whether vocalisers successfully communicated pain intensity to listeners, and which acoustic characteristics were responsible for variation in pain ratings. We found that the mean and range of voice fundamental frequency (F0, perceived as pitch), the amplitude of the vocalisation, the degree of periodicity of the vocalisation, and the proportion of the signal displaying nonlinear phenomena all increased with the level of simulated pain intensity. In turn, these parameters predicted increases in listeners' ratings of pain intensity. We also found that while different voice features contributed to increases in pain ratings within each level of expressed pain, a combination of these features explained an impressive amount of the variance in listeners' pain ratings, both across (76%) and within (31-54%) pain levels. Our results show that adult vocalisers can volitionally simulate and modulate pain vocalisations to influence listeners' perceptions of pain in a manner consistent with authentic human infant and nonhuman mammal pain vocalisations, and highlight potential for the development of a practical quantitative tool to improve pain assessment in populations unable to self-report their subjective pain experience.

Keywords: pain, nonverbal vocalisations, vocal communication, volitional

Introduction

Mammal vocal signals communicate key indexical information that is relevant in social and competitive contexts (Briefer, 2012; A. M. Taylor, Charlton, & Reby, 2016), and is highly conserved across species, (Owren, 2011; A. M. Taylor et al., 2016) including humans (e.g. Feinberg, Jones, Little, Burt, & Perrett, 2005; Koutseff et al., 2017; Puts, Hodges, Cárdenas, & Gaulin, 2007; Rendall, Vokey, & Nemeth, 2007; Sell et al., 2010). When experiencing pain, human infants (Bellieni, 2012; Levine & Gordon, 1982), human adults (Baker & Kenner, 1993; Fuller, Roberts, & McKay, 1993) and many nonhuman mammals (Bars, Gozariu, & Cadden, 2001; Bufalari, Adami, Angeli, & Short, 2007; Dubner, 1994; Mogil, 2009) produce pain vocalisations in response to noxious stimuli, that are in turn detected and processed via similar mechanisms in humans and nonhuman mammals (Schnitzler & Ploner, 2000; Tracey & Mantyh, 2007; X. J. Zhang, Zhang, Hu, & Xu, 2011). Vocal communication of pain is likely to provide survival advantages to signallers by attracting attention, aid, or protection (Craig, 2009; Levine & Gordon, 1982; Sullivan, 2008; Williams, 2002), and may also be advantageous to friendly receivers (warning of threat and danger, Craig, 2009; Sullivan, 2008; Williams, 2002). As such, pain vocalisations are likely to have been selected to communicate honest cues to pain levels in their acoustic structure (Hadjistavropoulos & Craig, 2002).

While multiple studies have reported differences in the occurrence, acoustic characteristics and perceptual characteristics of human infant and nonhuman mammal cries produced in response to pain versus other stressors, such as hunger or isolation (e.g. Boero, Bianchi, Volpe, Marcello, & Lenti, 1998; Calvino, Besson, Boehrer, & Depaulis, 1996; Fuller, 1991; Lindová, Špinka, & Nováková, 2015; Marx, Horn,

Thielebein, Knubel, & von Borell, 2003; Watts & Stookey, 1999; Weary, Braithwaite, & Fraser, 1998), ethical considerations limit the degree to which the graded acoustic communication of pain intensity can be investigated. Thus, most research in this area takes advantage of painful procedures already performed for purposes other than scientific investigation (e.g. medical: Facchini, Bellieni, Marchettini, Pulselli, & Tiezzi, 2005; Koutseff et al., 2017; agricultural: Puppe, Schön, Tuchscherer, & Manteuffel, 2005; White et al., 1995).

As human infants experience increases in pain, they produce cries with higher levels of roughness (irregular/chaotic vocal fold vibration) (Facchini et al., 2005; Koutseff et al., 2017; Tiezzi, Pulselli, & Facchini, 2004), higher amplitude (Fuller & Conner, 1995; Lehr et al., 2007; c.f. Maitre et al., 2017), lower variation in amplitude (Bellieni, Sisto, Cordelli, & Buonocore, 2004), longer bout duration (Johnston & O'Shaughnessy, 1987; Porter, Miller, & Marshall, 1986), and a more variable fundamental frequency, F0 (Koutseff et al., 2017; Porter et al., 1986). Mean F0 (perceived as pitch) appears not to correlate linearly with pain levels in infant cries (Johnston & O'Shaughnessy, 1987; Koutseff et al., 2017; Silva et al., 2010; c.f. Porter et al., 1986), but rather increases abruptly after a certain threshold of high pain is reached ("alarm threshold", Bellieni et al., 2004).

Acoustic cues to pain in nonhuman mammals have received relatively little consideration, and research has tended to focus disproportionately on calling rate (e.g. Kurejova et al., 2010; A. A. Taylor & Weary, 2000) rather than on variation in the spectral characteristics of calls. However, several studies have shown that call duration, amplitude and acoustic nonlinearities (irregular vocal fold vibration regimes, Fitch, Neubauer, & Herzel, 2002) increase with the intensity of electrical stimulation in mice (Eschaliere, Marty, Trolese, Moncharmont, & Fialip, 1988; Jourdan, Ardid, Chapuy,

Eschaliér, & Le Bars, 1995; Levine, Feldmesser, Tecott, Gordon, & Izdebski, 1984). In pigs, more painful castration procedures also induce vocalisations with higher peak frequencies (White et al., 1995), indicating cross-specific commonalities in the acoustic gradation of pain intensity.

Acoustic correlates of pain also co-vary with arousal in human speech (see Briefer, 2012), nonhuman mammal vocalisations (see Blumstein & Chi, 2012; Briefer, 2012), and human nonverbal vocalisations (Lima, Castro, & Scott, 2013; Nwokah, Davies, Islam, Hsu, & Fogel, 1993; Sauter, Eisner, Calder, & Scott, 2010; Szameitat, Darwin, Wildgruber, Alter, & Szameitat, 2011). This is because activation of the autonomic nervous system – which occurs when experiencing either pain (Benarroch, 2006) or arousal (Briefer, 2012) – affects respiratory and phonatory aspects of voice production (Briefer, 2012). Indeed, pain cries are assumed to exhibit higher F0 compared to distress cries caused by other stressors because they reflect a more highly aroused state (Boero et al., 1998; Fuller & Horii, 1986, 1988; Grunau, Johnston, & Craig, 1990; Gustafson & Harris, 1990; Johnston & O'Shaughnessy, 1987; Lingle, Wyman, Kotrba, Teichroeb, & Romanow, 2012).

Human listeners are able to distinguish infant pain cries from distress cries produced in response to other stressors (Gustafson & Harris, 1990; Koutseff et al., 2017; Porter et al., 1986; but see Lindová et al., 2015), and can discriminate more invasive from less invasive surgical circumcision procedures (Porter et al., 1986), likely as a result of graded differences in arousal rather than discrete, contextually discriminable acoustic characteristics. However, listeners cannot reliably distinguish between pain levels elicited by different vaccines, even though acoustic analyses reveal that more painful vaccines elicit cries with greater roughness (Koutseff et al., 2017). The few studies investigating perception of pain intensity suggest that higher-pitched

(Craig, Grunau, & Aquan-Assee, 1988; Porter et al., 1986), louder, and noisier (Porter et al., 1986) cries tend to be judged as more painful or urgent. In distress cries associated with other stressors (e.g. hunger, isolation), increased F0, F0 variability, duration, and roughness predict humans' perceptions of the urgency or level of distress experienced by human infants (Dessureau, Kurowski, & Thompson, 1998; Esposito, Nakazawa, Venuti, & Bornstein, 2012, 2015; Out, Pieper, Bakermans-Kranenburg, Zeskind, & van IJzendoorn, 2010; Reby, Levréro, Gustafsson, & Mathevon, 2016; Wood, 2009; Zanchi et al., 2016; Zeifman, 2004; see LaGasse, Neal, & Lester, 2005 for review) and infants of other primate species (F0 only, Kelly et al., 2017). Similarly, noisier (rhesus macaques: Jovanovic & Gouzoules, 2001) and higher frequency (pigs: Weary, Lawson, & Thompson, 1996) cries provoke more urgent responses in other mammals.

While in adult humans, the experience of pain can be reported verbally (X. J. Zhang et al., 2011), pain is also frequently expressed with nonverbal cries or screams, for example as a consequence of high-intensity pain (e.g. during childbirth, Fuller et al., 1993). Vocalisations are also considered valuable indicators of pain in groups unable to submit reliable self-reports regarding their subjective pain experience, such as older adults with advanced dementia, persons with intellectual disabilities, and patients at the end of life (Carter, McArthur, & Cunliffe, 2002; Herr, Coyne, McCaffery, Manworren, & Merkel, 2011; McGrath, Rosmus, Canfield, Campbell, & Hennigar, 1998; van Iersel, Timmerman, & Mullie, 2006). However, the acoustic structure of adult nonverbal pain vocalisations, and their effects on listeners' perceptions, have not yet been systematically investigated.

Here, to investigate the communication of pain in adult human vocalisations, trained actors were asked to produce pain vocalisations in three simulated contexts of

increasing pain intensity. Using acoustic analysis, we examined how simulated pain levels were encoded in the acoustic structure of these vocalisations. We then asked listeners to rate the pain levels experienced by the vocalisers, to test whether listeners correctly judged higher-intensity pain vocalisations as expressing more pain, and which acoustic characteristics affected their judgments. Given the apparent evolutionary continuity between other kinds of vocalisations produced by adult humans, infants and other mammals (Burling, 1993; laughter: Davila-Ross, Owren, & Zimmermann, 2009, 2010; Pisanski, Cartei, McGettigan, Raine, & Reby, 2016; infant distress cries: Lingle & Riede, 2014; Lingle et al., 2012; Zeifman, 2001), we predicted that acoustic encoding and perception of pain levels in adult simulated pain vocalisations would follow similar patterns to those observed in human infant and nonhuman mammal pain cries.

Method

1. Acoustic Analysis

Participants

We audio recorded 30 male and 30 female students of drama or acting from the Royal Central School of Speech and Drama and the University of Sussex, who received monetary compensation in exchange for their participation. All participants provided informed consent. None were currently suffering from any conditions that might affect their voice (e.g. cold, sore throat). This experiment was approved by the University of Sussex's Life Sciences & Psychology Cluster-based Research Ethics Committee (C-REC certificate of approval ER/JR307/4) and complies with the American Psychological Association's Ethical Principles of Psychologists and Code of Conduct.

Procedure

Voice recording. Vocalisations were recorded in a quiet room, with vocalisers standing 150 cm from a Zoom H4n microphone. A chair was positioned between the vocaliser and the microphone to restrict forward movement. Vocalisers were asked to imagine themselves in three painful situations of increasing intensity, and to produce a vocalisation in response to each imagined scenario. A description of each context was dictated by the experimenter and also displayed on a computer screen. The descriptions for each pain context were as follows:

Mild: Imagine you are experiencing a mild pain, one that is noticeable but manageable. Scalding your finger with boiling water or stubbing your toe are examples of this level of pain.

Moderate: Imagine you are experiencing a strong pain, one that is serious but not life-threatening. Examples of this level of pain are breaking your arm or dislocating your shoulder. Produce a vocalisation to express your pain.

Severe: Imagine you are experiencing the most intense pain you can think of. Examples are childbirth, or a life-threatening injury. Produce a vocalisation to express your pain.

In order to obtain realistic vocal stimuli, participants were encouraged to take as much time as they needed to immerse themselves in each imagined context, and to ‘let go of their inhibitions’. Participants were also given the option not to vocalise if they felt that they could not naturally produce the sentence or nonverbal vocalisation, and to repeat any sentence or vocalisation until they were satisfied with their portrayal. Recordings were saved as WAV files at 44.1 kHz sampling frequency and 16 bits amplitude resolution.

Spectral analysis. A total of 180 voice recordings (3 levels of pain intensity x 60 vocalisers) were acoustically analysed using a dedicated batch-processing script in PRAAT 5.3.62 DSP package (Boersma & Weenink, 2017). The script contained three distinct procedures. The first procedure characterized fundamental frequency (F0) and modulation (F0 contour variation). The F0 contour was extracted using the *To Pitch (cc)...* command. We systematically inspected each extracted pitch contour and verified it using a narrow band spectrogram displaying the first 2000 Hz of the signal. Erroneous

pitch values (e.g. octave jumps) were manually corrected by selecting the appropriate F0 candidate values in the edited pitch object. In segments displaying subharmonics (the presence of vocal fold vibration at a frequency equal to an integer multiple of the F0 in addition to the F0 itself, Fitch et al., 2002), the F0 was systematically preferred over the subharmonic. Where amplitude modulation (a subcategory of biphonation, whereby the air displacements of two independent sources of vocal energy, one of low frequency and one of higher frequency, interact to produce a signal with audible periodic variation in overall intensity, Fitch et al., 2002) was present, F0 values were selected only if clearly visible and audible. For segments where deterministic chaos (aperiodic, irregular vocal fold vibration, Fitch et al., 2002) was present, the automatically extracted pitch contour generally did not select F0 values; where it did, we manually deselected these values.

The F0 contour was used to derive the following parameters: mean F0, max F0, min F0, range F0, and F0CV (coefficient of variation in F0 across the entire duration of the signal). During inspection of each spectrogram, we also measured the proportion of the signal for which nonlinear phenomena (amplitude modulation, subharmonics, or deterministic chaos) were present, and created a measure representing this proportion as a percentage (%NLP).

Next, two distinct smoothing algorithms (*Smooth...* command in Praat) were performed on the pitch contour: the first (*Smooth...* command parameter = 25), suppressed period-to-period frequency fluctuations while preserving short-term, minor modulation events (such as vibrato-like frequency modulation, Charlton, Taylor, & Reby, 2017). The second (*Smooth...* command parameter = 2) suppressed short-term modulation, characterising only major F0 modulation events. After each smoothing procedure, inflection points were counted as each change in the sign of the contour's derivative, and divided by the total duration of the voiced segments in each recording.

This resulted in two distinct indexes of F0 modulation (inflex25 - minor inflections, and inflex2 - major inflections).

A second procedure characterised the mean amplitude of the stimuli, as well as amplitude range (intRange) and variability (intCV, the coefficient of variation of the intensity contour estimated using the *To intensity ...* command in PRAAT). A third procedure focused on the periodic quality of the signal and measured harmonics-to-noise ratio (HNR, a measure of the ratio of periodic components to non-periodic components), and two measures of F0 disturbance in the voiced proportion of the signal: jitter (small fluctuations in periodicity measured as the average of 'local', 'rap' and 'ppq5' measures in PRAAT) and shimmer (small variation in amplitude between consecutive periods, measured as the average of 'local', 'apq5' and 'apq11' parameters in PRAAT). Together, HNR, jitter and shimmer represent the overall 'degree of acoustic periodicity' of signals. Acoustic analysis procedures similar to these have been applied successfully in previous studies of human nonverbal vocalisations (e.g. babies' cries, Koutseff et al., 2017; Reby et al., 2016).

Statistical Analysis

Principal component analysis. To reduce our set of correlated acoustic variables to a smaller number of uncorrelated factors, we performed a principal component analysis (PCA) with varimax rotation on all aforementioned acoustic variables extracted from the full dataset of 180 vocalisations (Abdi & Williams, 2010) (see Table 3 for mean \pm SDs of these variables for each pain intensity level). We entered within-sex z-scores in place of raw measures for sexually dimorphic acoustic characteristics (mean F0, max F0, min F0, range F0).

Discriminant function analysis. To examine acoustic differences between pain intensities, we conducted a conventional leave-one-out DFA with forced entry (which is less vulnerable to collinear variables, random effects, and type I errors than stepwise entry, Mundry & Sommer, 2007) of the four principal components produced from the acoustic variables. We also conducted a MANOVA to establish whether there were significant differences in each raw acoustic variable between simulated pain intensity levels.

While duration was excluded from the PCA, DFA and MANOVA in order to focus these analyses on spectral variables, a separate repeated measures ANOVA was performed to test for differences in duration between the three levels.

2. Playback Experiment

Participants

Thirty females and 34 males (M age = 35.65 ± 9.53) from the USA were recruited using Amazon Mechanical Turk to provide voice-based assessments of the 180 previously acquired pain vocalisations (60 vocalisers x 3 vocalisations).

Participants completed the experiment using a custom computer interface designed and run on Synaesthesia Toolkit (Simner, 2018). All participants provided informed consent and were compensated with \$4 USD.

In order to reliably assess the effect of amplitude on listeners' attributions, it was necessary for listeners to maintain the same volume for the duration of the playback experiment. Eight participants who reported that they adjusted their volume settings were excluded from analyses.

This experiment was approved by the University of Sussex's Life Sciences & Psychology Cluster-based Research Ethics Committee (C-REC) (Certificate of approval: ER/JR307/8) and complies with the American Psychological Association's Ethical Principles of Psychologists and Code of Conduct.

Playback stimuli

Listeners rated all 180 voice stimuli acquired from the 30 male and 30 female acting students (60 vocalisers each producing three vocalisations corresponding to each level of simulated pain: mild, moderate and severe). These were the same vocal stimuli on which we performed acoustic analyses. Acoustic characteristics of these stimuli are described in the Results section.

Procedure

Listeners were instructed to use headphones and complete the experiment in a quiet place. To allow listeners to complete the experiment at a comfortable but audible volume, they were instructed to first set their volume to its lowest level. Listeners then heard a demo sound file (amalgamating a loud and a quiet stimulus), and were instructed to raise their volume until they could clearly hear the quiet vocalisation, while the loud vocalisation did not cause discomfort. Following this, listeners were asked not to adjust their volume settings during the experiment unless it became too uncomfortable. Listeners were asked at the end of the experiment if they adjusted their volume at any point. Due to the agonistic nature of the stimuli, listeners were made aware that if they felt uncomfortable or distressed listening to the sounds, they could stop the experiment.

Voice stimuli were blocked by sex. The order of blocks and stimuli within blocks was randomised. Before each block, participants were reminded to listen to each stimulus in full, and informed that they could take a break at any time. Listeners were instructed to, “Rate how much pain this vocalisation is conveying” on a 101-point Likert scale from 0 (no pain) to 100 (extreme pain).

Listeners were debriefed upon completing the study. They were told that the pain vocalisations were simulated, and that the vocalisers were not really experiencing pain. We examined reaction times against stimulus durations to ensure that participants completed the experiments properly. No participants were removed as a result of this process.

Statistical Analysis

We conducted a linear model testing the effects of intensity level, the four acoustic principal components, and duration on listeners’ pain ratings. The model included main effects and 2-way interactions between each of the four principal components and duration, and pain intensity level. We allowed the slopes of the relationship between pain ratings and the predictors to vary between both vocalisers and listeners, and tested null hypotheses based on the average of these slopes. The model included listener ID as a random subject variable, and vocaliser ID as a random factor. Effect sizes (provided in the Figures) were estimated using R^2 coefficients derived from simple linear regressions among relevant variables.

Results

Does the acoustic structure of simulated pain vocalisations differ with pain intensity?

Principal component analysis. This unsupervised analysis produced four components with eigenvalues greater than 1 (Kaiser's criterion). These components explained 33%, 21%, 14%, and 10% of the variance in acoustic characteristics, respectively. Acoustic variable loadings on the components are reported in Table 1.

Variable loadings indicated that the first principal component (PC1) indexed the degree of periodicity of the vocalisation and the F0 modulation of its voiced proportion: vocalisations with higher PC1 scores were more periodic, had a lower level of jitter and shimmer, and had more minor (short-term) and major (longer-term) F0 inflections. Vocalisations with higher PC2 values had a higher mean amplitude, a higher minimum, mean, and maximum F0, and displayed more nonlinear phenomena. PC2 can reasonably be interpreted as an index of subglottal pressure. Indeed, amplitude and F0 both increase with subglottal pressure, as increasing pressure below the glottis raises both the speed at which the vocal folds vibrate and the energy imparted to displaced air upon vocal fold opening (Behrman, 2007; Herbst, 2016); nonlinearities are also observed at the upper limits of subglottal pressure (Berry, Herzel, Titze, & Story, 1996; Fitch et al., 2002; Herbst, 2016; Jiang, Zhang, & Stern, 2001; Y. Zhang & Jiang, 2005). PC3 characterised the range of F0, primarily driven by high maximum F0 values (resulting in higher F0 range, and higher F0CV). The final component (PC4) indexed amplitude variability: vocalisations with higher PC4 scores had higher intCV and intRange values.

Discriminant function analysis. Discriminant function analysis indicated that the three pain intensities were acoustically distinct (Figure 2): the classification success rate was significantly greater than chance (correct classification percentage = 75.6%, chance = 33.33%, $p < .0005$). Table 2 reports the loadings of the acoustic principal components

on the first three discriminant functions. The first discriminant function (eigenvalue = 1.82, variance explained = 96.8%) was the key differentiator of intensity categories (Figure 2), demonstrating that the degree of periodicity of the signal and the F0 modulation of its voiced proportion (PC1), F0, amplitude, nonlinear phenomena (PC2), and F0 variation (PC3) all increased with pain intensity. The second discriminant function (eigenvalue = 0.06, variance explained = 3.2%) was not important in discriminating groups.

Using Pillai's trace, a MANOVA revealed that there was a significant effect of pain intensity on the raw acoustic variables ($V = 8.75$, $F(28, 330) = 8.75$, $p < .0005$). Separate univariate ANOVAs revealed that the effect of pain intensity was significant for each acoustic variable (all $ps < .012$). Tables 3 and 4 report the mean values of the raw acoustic variables, as well as the principal components, for each vocaliser sex and pain intensity level. Patterns of acoustic variation were comparable across sexes.

A repeated measures ANOVA revealed that duration also differed between levels, $F(2,177) = 41.7$, $p < .0005$. Paired comparisons (LSD) showed that mild pain vocalisations ($0.68 \pm 0.23s$) were significantly shorter than moderate pain vocalisations ($1.77 \pm 0.23s$), and that both mild and moderate vocalisations were significantly shorter than severe pain vocalisations ($3.64 \pm 0.23s$, all $ps < 0.002$).

Do pain intensity level and acoustic characteristics affect ratings of pain?

Linear mixed model analysis revealed a significant effect of pain intensity level on pain ratings (Table 5): mild intensity pain vocalisations were rated as conveying the least pain ($M = 16.61 \pm 1.31$), followed by moderate intensity vocalisations ($M = 44.21 \pm 1.19$), with severe intensity vocalisations rated as conveying the most pain ($M = 75.25 \pm 1.20$).

All four principal components and duration significantly predicted pain ratings (Table 5). Higher pain ratings were associated with longer duration, greater periodicity and F0 modulation (PC1), higher F0, amplitude, and nonlinear phenomena (PC2), greater F0 variation (PC3), and greater intensity variation (PC4), although the effect size for PC4 was minimal (Figure 3).

As illustrated in Figure 3, pain ratings increased as principal component values increased. However, the relative contribution of individual principal components in predicting listeners' ratings of pain intensity differed across intensity levels. Variation in PC1 had the greatest effect on pain ratings in moderate pain vocalisations, a smaller effect in mild pain vocalisations, and no effect in severe pain vocalisations. The effect of PC1 on pain ratings was also more reliable in moderate than in mild pain vocalisations. PC2 only reliably affected pain ratings within the mild intensity category. Listeners were sensitive to variation in PC3 only in severe and moderate pain vocalisations, but the effect of PC3 on pain ratings was much more reliable in severe pain vocalisations. Finally, PC4 increased marginally with pain ratings within moderate pain vocalisations, but effect sizes were minimal. These results demonstrate that acoustic variables contribute differently to listeners' perceptions of pain at different pain intensity levels.

Inspection of spectrograms (see examples in Figure 1) suggested that vocalisations often transitioned between highly periodic (PC1) and highly chaotic (PC2) regimes of vocal fold vibration (e.g. Figure 1 spectrograms 4 and 6). Vocalisations with such bifurcations would not score highly on individual components, despite exhibiting multiple characteristics associated with high pain ratings. In addition, some vocalisations exhibited octave jumps or other forms of F0 variation (producing high PC3 scores) concurrently with periodic or chaotic vibratory regimes (e.g. Figure 1 spectrogram 6), of which the possible additive effect on pain ratings cannot be assessed by testing each PC individually.

Therefore, for each vocalisation, we computed an average of values for the first three principal components (excluding PC4 due to the observed minimal effect sizes), and conducted a fully factorial linear mixed model with only pain intensity and the average of PCs 1-3 (PC123) as predictors. Both main effects and interaction terms were highly significant (all F s > 132.36, all p s < .001). Higher pain ratings were associated with higher PC123 scores, with PC123 explaining 75.5% of the variance in listeners' pain ratings (Figure 4). Within pain intensity levels, variation in PC123 had the greatest effect on pain ratings in moderate pain vocalisations.

Discussion

Our results show that acoustic variation in simulated pain vocalisations produced by adult men and women is organised along three main groups of acoustic characteristics. Together, these acoustic components are sufficient to reliably separate vocalisations by their simulated level of pain intensity (mild, moderate and severe) and in turn reliably predict assessments of pain intensity by adult listeners. At the same

time, the relatively continuous distribution of both acoustic characteristics and listeners' pain ratings between and within these categorical levels suggests that, overall, simulated pain was communicated in a graded manner. Moreover, while the relative contribution of acoustic characteristics to listeners' pain ratings varied within each level of simulated pain intensity, their combination (by averaging) was the strongest and most reliable predictor of listeners' pain ratings across and within pain levels.

Acoustic cues to levels of pain intensity

The results of the principal component analysis revealed that the acoustic variability of simulated pain vocalisations could be described by three uncorrelated groups of acoustic variables. A first group of variables (all loading on PC1) characterised the degree of periodicity (HNR, jitter and shimmer) of the signal and the F0 modulation (rate of short term and long-term inflections) of its voiced proportion. A second group of variables (loading on PC2) characterised the pitch (min and mean F0), amplitude (mean amplitude) and occurrence of nonlinear phenomena (percentage presence – %NLP), which are all known to increase with subglottal pressure. Finally, a third group of variables (max and range F0, F0 CV, all loading on PC3) represented pitch range and variability. The fact that the vocalisations were clearly organised according to the three increasing levels of simulated pain intensity in the three-dimensional space created by these components strongly indicates that a large proportion of the acoustic variation in these vocalisations served to express pain intensity (see Figures 1 & 3).

These results support our predictions, which stem from previous work on human infants and other mammals. Indeed, indicators of subglottal pressure and pitch range have previously been shown to encode pain intensity in pain vocalisations produced by

human infants (F0: Bellieni et al., 2004; roughness: Facchini et al., 2005; Koutseff et al., 2017; Tiezzi et al., 2004; amplitude: Fuller & Conner, 1995; Lehr et al., 2007; F0 variability/range: Koutseff et al., 2017; Porter et al., 1986) and nonhuman mammals (F0: White et al., 1995; roughness: Levine et al., 1984; amplitude: Eschali r et al., 1988; Jourdan et al., 1995). These acoustic features also influence perceived urgency of caregivers in nonhuman mammals (Jovanovic & Gouzoules, 2001; Weary et al., 1996), as well as assessments of pain (Craig et al., 1988; Porter et al., 1986) or distress (Esposito et al., 2015; Kelly et al., 2017; LaGasse et al., 2005; Wood, 2009) in adult humans listening to infant distress cries. Thus, the observed increases in acoustic indicators of subglottal pressure and pitch range, as a function of simulated and perceived pain intensity level, are consistent with acoustic mediators of pain communication observed in authentic pain vocalisations produced by human infants and infant or adult nonhuman mammals.

We also found that, as vocalisers simulated higher pain levels, they produced vocalisations with more modulated F0 (short- and long-term inflections, contributing to PC1). To our knowledge, this is the first time that frequency modulation has been identified as communicating pain intensity, although high frequency modulation is associated with calls produced in fearful contexts in nonhuman mammals (Briefer, 2012). Inspection of spectrograms suggested that vocalisations with high short-term F0 modulation were either characterised by vibrato-like frequency modulation (Figure 1, spectrograms 2, 3, 4 and 5), and/or numerous glottal stops (Figure 1, spectrograms, 1 4, 5, and 6), both giving the vocalisations a cry/sob-like quality. Similar shifts in vocal quality have been observed in infant cries, where individual cries within bouts become shorter and more frequent as pain increases (Porter et al., 1986).

While the occurrence of nonlinear phenomena (contributing to PC2) increased as levels of simulated pain intensity increased, the degree of periodicity, driven by the voiced proportion of the signal (i.e. the proportion with a detectable pitch, loading on PC1) increased, thus contrasting previous research on human infant pain cries (Koutseff et al., 2017). We argue that this is primarily driven by the breathy voice quality that characterised the majority of mild intensity vocalisations (see Figure 1, spectrograms 7 & 8), but is not observed in infant cries (Facchini et al., 2005; Koutseff et al., 2017). Breathy voice is produced with minimal glottal closure (Gobl & Chasaide, 1992), resulting in turbulent airflow accompanying vocal fold vibration and therefore producing a much less periodic acoustic signal than modal speech (de Krom, 1995; Gobl & Chasaide, 1992; Herbst, 2016; Hillenbrand, 1988; Hillenbrand, Cleveland, & Erickson, 1994; Hillenbrand & Houde, 1996; Scherer, 1986) or shouted speech (C. Zhang & Hansen, 2007). In contrast, the higher amplitude of moderate and severe intensity vocalisations is associated with greater and more abrupt glottal closure (Backstrom, Alku, & Vilkman, 2002; Södersten, Hertegård, & Hammarberg, 1995), achieved through high vocal fold tension and resulting in relatively less turbulent air leakage (associated with “pressed” voice quality, Gobl & Chasaide, 1992; Herbst, 2016; Södersten et al., 1995), and therefore a more periodic acoustic signal.

However, as subglottal pressure reaches the upper limits at which the vocal folds vibrate stably, the vocal folds transition to chaotic regimes of vibration (Fitch et al., 2002; Herbst, 2016; see Figure 1), which can overlay or replace periodic spectral components (as observed in infant cries, Facchini et al., 2005; Koutseff et al., 2017). Highly irregular, unvoiced portions in acoustic recordings (during which pitch is absent or undetectable) are not considered in jitter and shimmer measures, but are represented by the percentage of the signal for which nonlinear phenomena are present (contributing

to PC2). Thus, vocalisations may be characterised by either high PC1 values (highly periodic), high PC2 values (highly chaotic), or, where vocalisations transition between periodic and highly chaotic vocal regimes (bifurcations, Fitch et al., 2002; Herbst, 2016; e.g. Figure 1, spectrograms 4 & 6), a combination of the two. The prevalence of nonlinear phenomena in vocalisations associated with higher pain corroborates a growing body of evidence that this acoustic characteristic typically serves to attract attention (Arnal, Flinker, Kleinschmidt, Giraud, & Poeppel, 2015; Blumstein & Récapet, 2009; Charlton, Watchorn, & Whisson, 2017; Mitani & Stuht, 1998; Reby & Charlton, 2012).

A follow-up discriminant analysis based on the first three acoustic principal components reliably classified vocalisations according to the three levels of simulated pain intensity (76% correct classification). Should this high reliability extend to authentic pain vocalisations, our multivariate acoustic analyses may form the basis for the development of a practical quantitative tool to improve pain assessment in populations unable to self-report their subjective pain experience (Docking, Lane, & Schofield, 2017; Herr et al., 2011), especially as pain levels appear to be discriminated more sensitively by acoustic analysis than by perceptual judgments (Koutseff et al. 2017). Future research could apply this methodology to real pain vocalisations such as childbirth vocalisations (Fuller et al., 1993), wherein acoustic indicators of pain may offer a viable alternative (Baker & Kenner, 1993) to obtrusive and much-criticised vaginal examination (Dahlen, Downe, Duff, & Gyte, 2013; Shepherd & Cheyne, 2013) in monitoring labour stage.

Relationships between acoustic cues and listeners' assessments of pain intensity

The values of each principal component varied not only between, but also within pain intensity levels, and predicted pain ratings both within and across these levels relatively continuously, supporting the contention that acoustic communication of pain is graded (likely as a function of distress-related arousal), rather than discrete (Bellieni, 2012; Bellieni et al., 2004; Briefer, 2012; Kelly et al., 2017; Out et al., 2010; Porter et al., 1986; Sauter et al., 2010). Moreover, acoustic variation in our simulated pain vocalisations predicted listeners' perceptions of pain in a manner consistent with reported effects of F0, amplitude and roughness on the urgency of nonhuman mammals' responses to distress cries (Jovanovic & Gouzoules, 2001; Weary, Lawson, & Thompson, 1996), and on adult humans' assessments of pain in infant distress cries (Craig et al., 1988; Porter et al., 1986).

Interestingly, the relative contribution of each component to listeners' pain ratings varied within pain intensity levels. Mild intensity vocalisations tended to be characterised by shorter duration, indicators of low subglottal pressure, indicators of breathy voice quality, low F0 range, and elicited low pain ratings. Within this category, pain ratings were mainly driven by indicators of subglottal pressure (PC2), and to a lesser extent, breathiness (PC1). Moderate intensity vocalisations had intermediate duration and elicited higher pain ratings, but ratings were primarily influenced by the vocalisation's periodicity (i.e. the degree to which the vocalisation had a breathy (aperiodic) or pressed (periodic) voice quality) and pitch range (e.g. due to frequency jumps, high max F0). Finally, severe pain vocalisations were longer and tended to either be highly periodic, highly chaotic, or transitioned between the two vocal fold vibration regimes, and elicited the highest pain ratings. Yet regardless of vibratory regime, pitch range largely determined whether severe pain vocalisations were rated relatively low or high on pain level.

The increase in both degree of periodicity and nonlinear phenomena (characterised by PC1 and PC2 scores) with pain intensity and pain ratings suggests that pain can be communicated via distinct, seemingly opposing acoustic regimes (periodic vs. chaotic). Opposite relationships between roughness and distress-related arousal have also been documented in different species (Facchini et al., 2005; Levine & Gordon, 1982; Stoeger, Charlton, Kratochvil, & Fitch, 2011; c.f. Blumstein & Chi, 2012; Linhart, Ratcliffe, Reby, & Špinka, 2015; Puppe et al., 2005), and, in piglets, between call types in the same distress-inducing context (Linhart et al., 2015). Importantly, a combination of the first three acoustic principal components contributed substantially to the high accuracy of our discriminant analysis (76%), and more strongly and reliably predicted pain ratings both across ($R^2 = 76\%$) and within (R^2 s = 31-54%) intensity levels than did any individual acoustic component. Therefore, while pain can be conveyed via multiple acoustic routes, and the relative correlation between each individual acoustic component and pain ratings varies across pain levels, it is their additive presence that appears to most effectively communicate pain intensity.

Are simulated vocalisations functional?

The fact that we focused on simulated pain vocalisations may be seen as a limitation affecting the ecological relevance of our results. However, while there are acoustic, perceptual, and neural differences between simulated and authentic nonverbal vocalisations (Anikin & Lima, 2017; Bryant & Aktipis, 2014; Lavan, Scott, & McGettigan, 2015; McGettigan et al., 2015), acted portrayals are generally considered acceptably similar to spontaneous nonverbal vocalisations (Sauter et al., 2010; Sauter & Fischer, 2017). In particular, simulated pain vocalisations are among the most likely to be classified as authentic, and there is a smaller difference in listeners' judgments of

authenticity between spontaneous and simulated pain vocalisations than for most other vocalisations (Anikin & Lima, 2017). Consistent with this, we found that the expression and perception of pain in these vocalisations appeared to follow similar rules to those reported in the vocalisations of preverbal human infants and nonhuman mammals (as discussed in the previous sections). In particular, the substantially larger increase in F0 between moderate and severe intensities than between mild and moderate intensities that we report suggests that actors produced vocalisations mirroring the previously observed ‘alarm threshold’ in human infant pain cries (Bellieni et al., 2004), rather than capitalising on more linear associations between F0 and perceived pain (Craig et al., 1988; Porter et al., 1986) to influence listeners’ attributions.

Moreover, simulation is likely to be an integral component of the spontaneous communication of vocal pain in adult humans. Recent evidence that non-actors may provide vocal expressions as realistic as those produced by actors (Jürgens, Grass, Drolet, & Fischer, 2015) suggests that the capability to accurately simulate spontaneous vocalisations and elicit appropriate listener responses may not be limited to actors. Indeed, humans can even modulate (exaggerate or minimise) responses to genuine pain depending on context, mood, and cognition (see Tracey & Mantyh, 2007), indicating that spontaneous expression of pain is dependent not just on nociceptive input, but also on communicative intentions. Future work could investigate whether listeners can detect exaggeration in partially or fully simulated pain vocalisations.

Humans’ ability to modulate or simulate pain expression is also consistent with functional vocal deception in other social mammals, which is commonly observed in survival contexts despite the potential costs associated with ‘crying wolf’ (Oesch, 2016; Schmid, Karg, Perner, & Tomasello, 2017). For example, in capuchin monkeys, deceptive alarm calls are acoustically indistinguishable from predator-elicited alarm

calls, and evoke comparable responses from conspecifics (Wheeler & Hammerschmidt, 2013). Vocal pain exaggeration or simulation may thus be an adaptive survival-enhancing strategy, for example eliciting urgent aid. Such volitional modulation of nonverbal vocalisations may have been at the origins of selection for increased vocal control, eventually culminating in the emergence of articulated speech in humans (Oesch, 2016; Pisanski et al., 2016).

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Declaration of Interests

The authors report no competing interests.

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Table 1. Rotated factor loadings for each principal component calculated from the acoustic variables characterising simulated pain vocalisations. Percentage of explained variance in acoustic characteristics and eigenvalues for each factor are given below. The highest factor loading for each acoustic variable across PCs is highlighted in bold.

	PC1	PC2	PC3	PC4
	Variance = 33%	Variance = 21%	Variance = 14%	Variance = 10%
Acoustic variable	Eigenvalue = 4.57	Eigenvalue = 2.96	Eigenvalue = 1.97	Eigenvalue = 1.39
HNR (dB)	.92	.04	-.02	.04
Jitter (Hz)	-.79	.00	.08	-.15
Shimmer (dB)	-.76	-.04	.09	.00
Minor F0 inflections	.72	.17	.18	-.11
Major F0 inflections	.72	.03	.23	-.01
Minimum F0 (Hz)	.05	.91	-.11	.05
Mean F0 (Hz)	.10	.87	.31	.02
Mean amplitude (dB)	.37	.75	.22	-.02
Nonlinear phenomena (%)	-.34	.59	.11	-.30
F0 CV (Hz)	-.07	-.09	.95	.05
F0 range (Hz)	.11	.28	.93	.06
Max F0 (Hz)	.12	.56	.77	.08
Intensity CV (dB)	-.22	-.24	-.03	.92
Intensity range (dB)	.31	.25	.25	.81

Table 2. Principal component (PC) loadings on the discriminant functions (DF).
Principal components are defined in Table 1.

Acoustic variable	DF1	DF2
PC1 – Degree of periodicity	.48	.44
PC2 – F0, amplitude, nonlinear phenomena	.43	-.32
PC3 – F0 variation	.24	-.41
PC4 – Intensity variation	.06	.78

Table 3. Mean acoustic characteristics of female vocal stimuli. Figures in square brackets represent standard errors of the mean.

Acoustic variable	Pain intensity		
	Mild	Moderate	Severe
Mean F0 (Hz)	465.5 [43.31]	539.9 [35.57]	737.8 [55.28]
Max F0 (Hz)	537.4 [50.86]	697.4 [53.58]	983.6 [78.92]
Min F0 (Hz)	367.2 [37.07]	383.2 [17.41]	464.6 [34.41]
Range F0 (Hz)	170.2 [28.48]	314.2 [41.68]	519.0 [67.06]
F0 CV (Hz)	0.11 [0.02]	0.15 [0.02]	0.17 [0.03]
Minor F0 inflections	1.84 [0.25]	3.92 [0.42]	6.77 [0.65]
Major F0 inflections	0.19 [0.04]	0.41 [0.06]	0.64 [0.08]
Mean amplitude (dB)	55.10 [1.38]	62.70 [1.32]	71.38 [0.93]
Intensity range (dB)	22.74 [1.07]	29.54 [0.90]	31.26 [1.41]
Intensity CV (dB)	1.19 [0.06]	1.16 [0.06]	0.90 [0.07]
Shimmer (dB)	0.15 [0.01]	0.11 [0.01]	0.10 [0.01]
Jitter (Hz)	0.029 [0.002]	0.017 [0.002]	0.018 [0.002]
HNR (dB)	5.73 [0.99]	10.57 [0.92]	12.91 [1.09]
Nonlinear phenomena (%)	44.69 [6.34]	39.70 [4.55]	55.07 [5.05]
Principal component	Mild	Moderate	Severe
PC1 – Degree of periodicity	-0.49 [0.15]	0.44 [0.17]	0.92 [0.19]
PC2 – F0, amplitude, %NLP	-0.40 [0.19]	-0.21 [0.12]	0.49 [0.18]
PC3 – F0 variation	-0.42 [0.12]	-0.05 [0.14]	0.45 [0.23]
PC4 – Intensity variation	-0.26 [0.15]	0.01 [0.12]	-0.42 [0.18]

Table 4. Mean acoustic characteristics of male vocal stimuli. Figures in square brackets represent standard errors of the mean.

Acoustic variable	Pain intensity		
	Mild	Moderate	Severe
Mean F0 (Hz)	270.7 [12.54]	340.4 [9.38]	440.8 [20.84]
Max F0 (Hz)	312.3 [14.98]	420.4 [15.79]	654.9 [59.72]
Min F0 (Hz)	209.0 [11.19]	251.6 [10.07]	296.1 [12.85]
Range F0 (Hz)	103.3 [9.08]	168.8 [17.42]	358.8 [61.96]
F0 CV (Hz)	0.13 [0.01]	0.13 [0.01]	0.18 [0.03]
Minor F0 inflections	0.80 [0.15]	2.79 [0.37]	5.22 [0.52]
Major F0 inflections	0.12 [0.03]	0.27 [0.05]	0.49 [0.06]
Mean amplitude (dB)	52.33 [1.30]	62.62 [1.37]	69.99 [1.15]
Intensity range (dB)	22.33 [1.40]	32.42 [1.21]	37.19 [1.89]
Intensity CV (dB)	1.27 [0.07]	1.39 [0.07]	1.17 [0.10]
Shimmer (dB)	0.17 [0.01]	0.14 [0.01]	0.13 [0.01]
Jitter (Hz)	0.038 [0.003]	0.022 [0.001]	0.020 [0.002]
HNR (dB)	2.51 [0.44]	6.82 [0.49]	8.44 [0.76]
Nonlinear phenomena (%)	40.70 [3.94]	48.58 [5.35]	64.84 [4.58]
Principal component	Mild	Moderate	Severe
PC1 – Degree of periodicity	-0.96 [0.10]	-0.17 [0.10]	0.26 [0.14]
PC2 – F0, amplitude, %NLP	-0.74 [0.13]	0.03 [0.14]	0.83 [0.17]
PC3 – F0 variation	-0.22 [0.08]	-0.21 [0.11]	0.44 [0.28]
PC4 – Intensity variation	-0.20 [0.18]	0.52 [0.15]	0.35 [0.24]

Table 5. Linear mixed model testing the effects of the intensity of pain simulated by the vocaliser the four acoustic principal components, and duration on listeners' attributions of the level of pain conveyed by the vocaliser.

Source		<i>df</i>₁, <i>df</i>₂	<i>F</i>	<i>p</i>
i.	Intercept	1, 67.44	685.60	< .001
ii.	Pain intensity	2, 9928.98	538.71	< .001
iii.	PC1	1, 9929.04	457.73	< .001
iv.	PC2	1, 9929.00	1081.00	< .001
v.	PC3	1, 9928.98	219.46	< .001
vi.	PC4	1, 9929.00	31.56	< .001
vii.	Duration	1, 9928.98	232.82	< .001
viii.	Pain intensity * PC1	2, 9928.99	158.59	< .001
ix.	Pain intensity * PC2	2, 9928.98	105.63	< .001
x.	Pain intensity * PC3	2, 9928.99	55.31	< .001
xi.	Pain intensity * PC4	2, 9929.00	56.06	< .001
xii.	Pain intensity * Duration	2, 9928.99	18.97	< .001

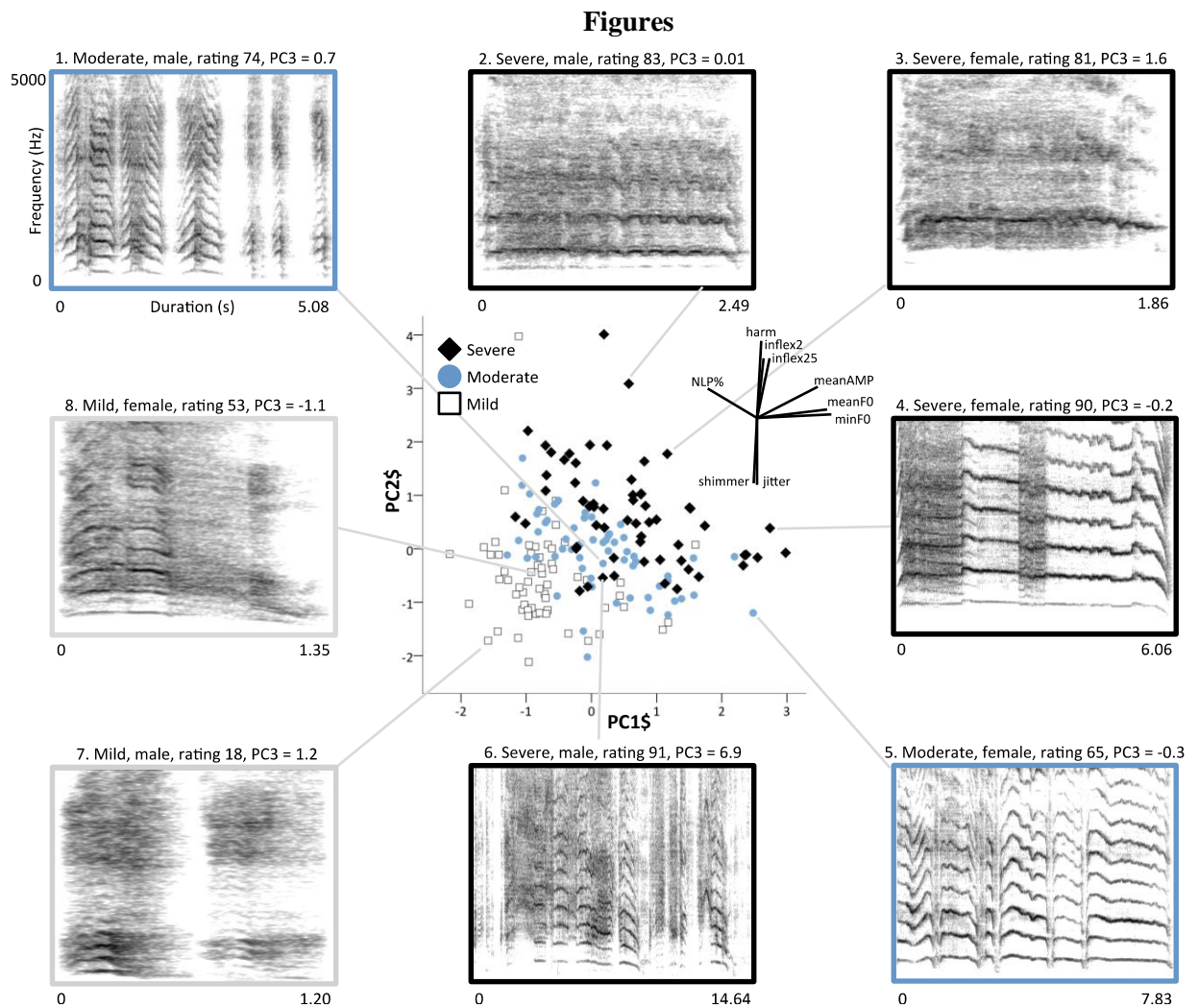


Figure 1. Principal component analysis (PCA) illustrating the acoustic variability of pain vocalisations across pain intensity levels. Each pain vocalisation is plotted against its score along the first two principal components. The radar plot in the top right corner of the scatterplot represents PC factor loadings of the acoustic variables. Spectrograms illustrate how the vocalisations vary along the principal components. The text directly above each spectrogram describes: the name of the corresponding audio file accessible in the Electronic Supplementary Materials, vocaliser sex, pain intensity, the mean pain rating attributed to the vocalisation, and score on the third principal component.

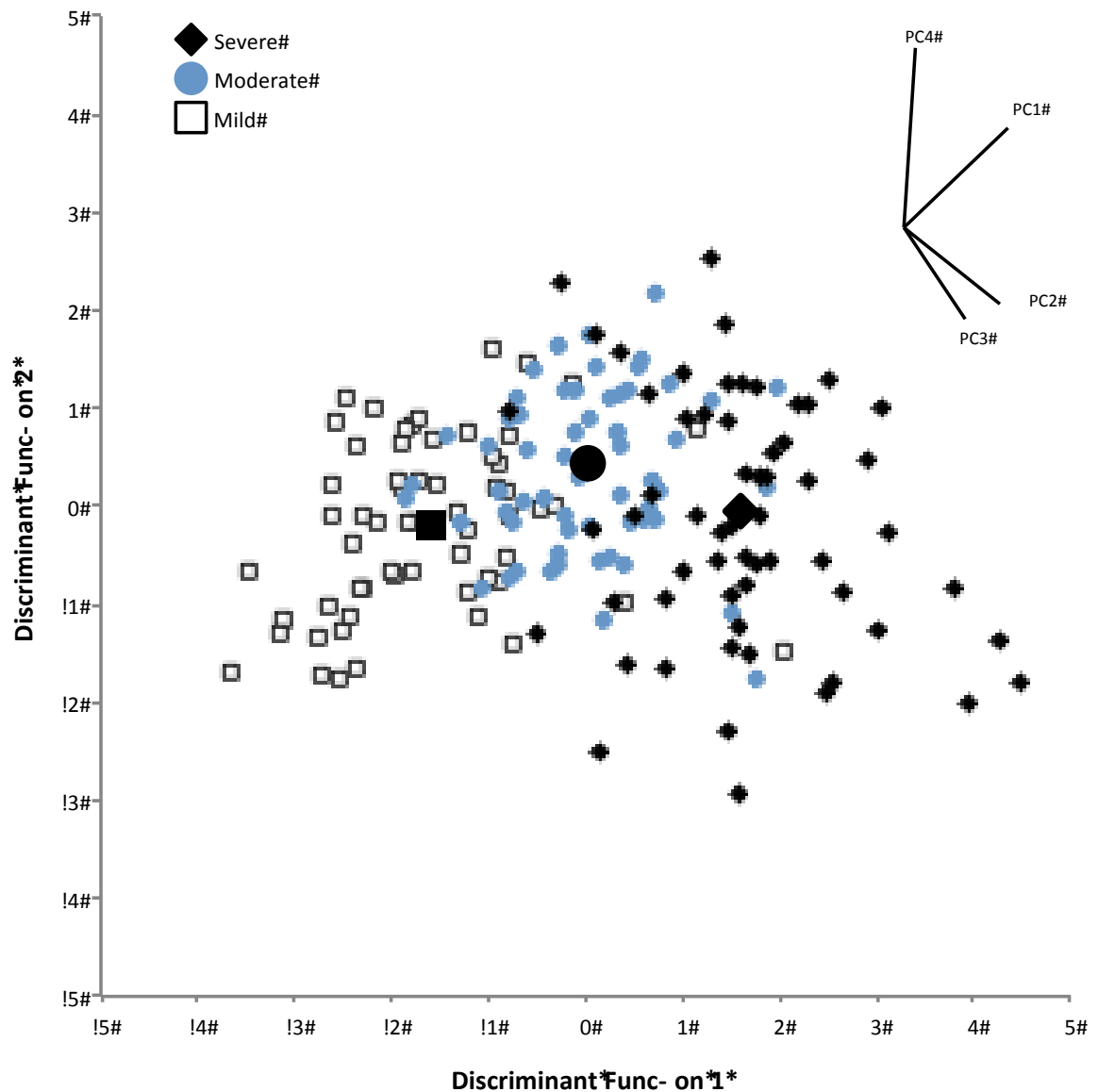


Figure 2. Discriminant function analysis illustrating acoustic separation of pain vocalisations at different levels of pain intensity. Each data point represents the centroid of a vocal stimulus as a function of the first two discriminant variables that maximise individual separation. Larger black data points represent mean group centroids for each stimulus condition. The radar plot on the top right represents the loadings of the principal components on the first two discriminant functions. Pain intensity categories were mainly separated on the first three principal components (see Table 1).

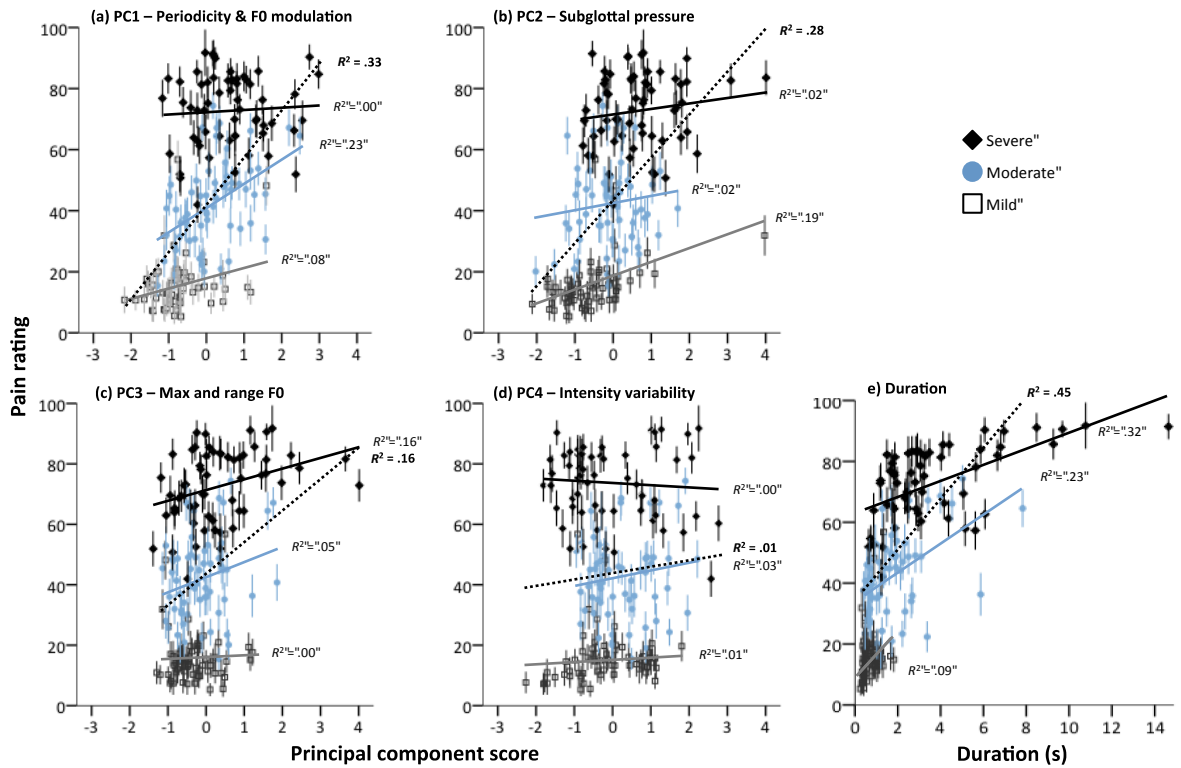


Figure 3. Pain rating as a function of variation in the four principal components (a)-(d) and stimulus duration (e). Each data point represents the mean pain rating averaged across listeners for each pain vocalisation. Error bars represent 95% confidence intervals. White squares represent mild pain simulations; blue circles represent moderate pain simulations; black diamonds represent severe pain simulations. R^2 values for each regression line (calculated based on mean pain ratings) are reported in the graphs. Dotted regression lines represent the overall regression (pooling across pain intensity levels). (a) PC1 represents the degree of periodicity of the signal and the F0 modulation of its voiced proportion. (b) PC2 represents indicators of subglottal pressure (mean amplitude, F0, and the proportion of the signal displaying nonlinear phenomena). (c) PC3 represents max and range F0. One severe intensity value (6.87, 91) is not represented in the graph but is included in the regression lines. (d) PC4 represents intensity variability.

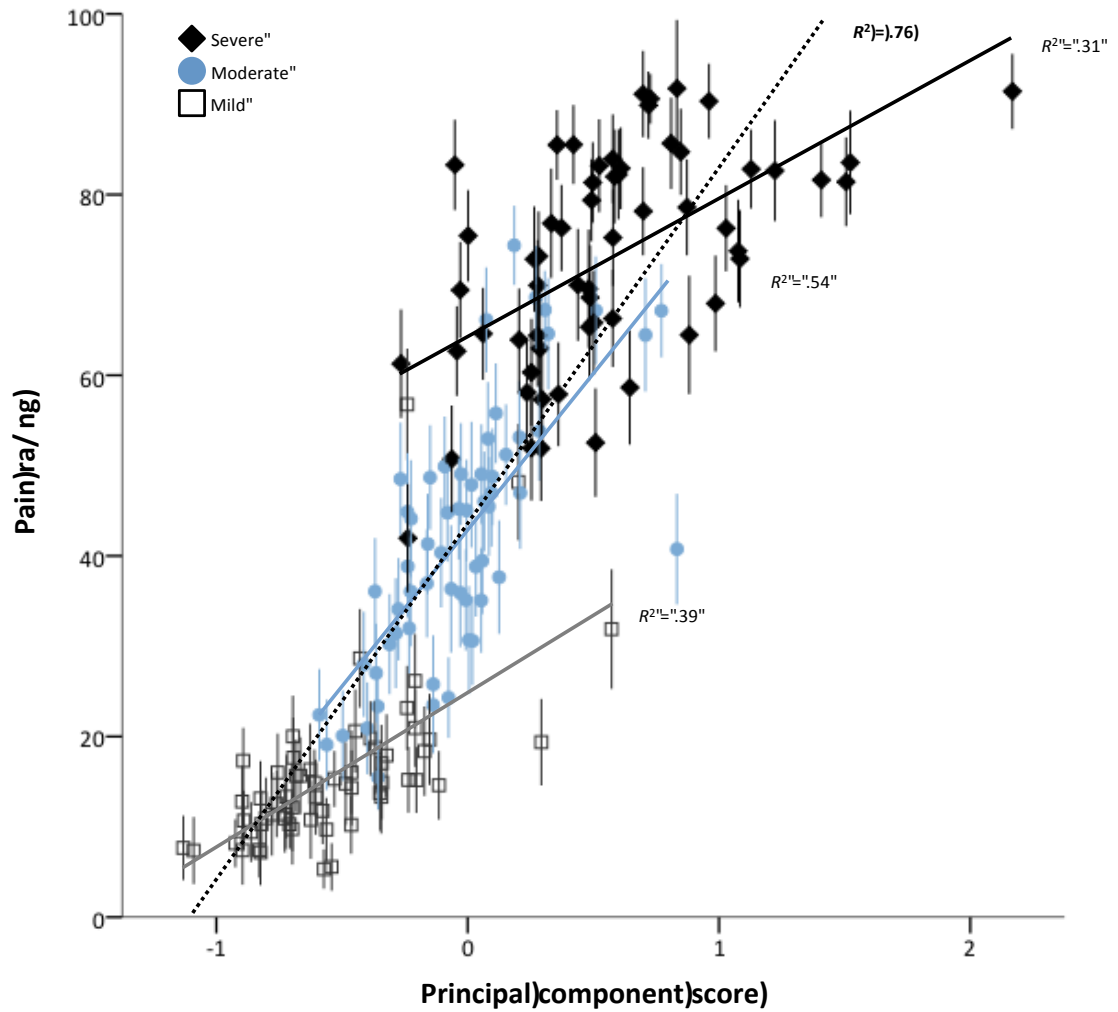


Figure 4. Pain ratings as a function of the average of the first three principal components. Each data point represents the mean pain rating averaged across listeners for each pain vocalisation. Error bars represent 95% confidence intervals. White squares represent mild pain simulations; blue circles represent moderate pain simulations; black diamonds represent severe pain simulations. R^2 values for each regression line (calculated based on mean pain ratings) are reported in the graphs. The dotted regression line represents the relationship between each principal component and pain ratings across pain intensity levels. This regression line explains the most (76%) variance in pain ratings.